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Importance of the Indoor Environment in Air Pollution Exposure

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ABSTRACT

A portable personal air pollution sampler was used to measure the exposure of twenty children to respirable particulates, sulfur dioxide, and nitrogen dioxide over a 24-hour period. Particulate exposures were significantly higher among children who lived with one or more smokers, and exceeded the primary air quality [illegible] of the twenty subjects. To a large extent, an individual's respirable particulate load appears to be determined by exposure to indoor rather than outdoor pollution.

AIR POLLUTION STUDIES of children have been of particular interest because children constitute a nonsmoking, nonoccupationally-exposed group who have generally lived in one community for most of their lives. To date, such investigations have concentrated on outdoor air pollution. However, children living in a temperate region spend from 60% to 80% of their time indoors on an average school day. We therefore decided to monitor the air pollution exposure of twenty boys continuously over a 24-hour period to determine the importance of the indoor environment as a determinant of an individual's total exposure to air pollution.

Some evidence suggests that indoor pollution may be related to childhood illness.[1,2] In addition, differences in respiratory symptoms and ventilatory function between black and white children,[3] and the intersubject variability in respiratory illness and lung function in any given community may be partially explained by differences in indoor pollutant exposures. We examined the relationship, if any, between personal pollutant load and respiratory disease.

Materials and Methods

From April through June 1973, 265 children (102 boys, 163 girls) aged 12 to 17 years were seen as part of a community respiratory disease survey in Ansonia, Connecticut.[4] From the 102 boys in the survey, 5 black and 5 white subjects with respiratory disease were randomly chosen, together with 5 black and 5 white normal control subjects matched for age within and between the racial groups. All subjects were nonsmokers and had lived in the area for at least the preceding 5 years. Criteria for selection in the disease groups were: 1) cough on most days, 2) sputum production on most days, 3) wheezing at least once a month, and/or 4) a history of asthma. The presence of respiratory disease was corroborated by pulmonary function tests.

The personal air sampling equipment was contained in a 56 cm by 41 cm by 15 cm (22 in by 16 in by 6 in) suitcase weighing 10 kg (22 lbs). This sampler could be carried by a subject wherever he went and positioned to take in air at breathing level. Based on a system developed by Burgess and associates,[5] the sampler was modified in this laboratory to sample continuously for 24 hours, without significant fluctuations in air flow rates, after having been connected to an AC power supply for the previous 16 hours. Three separate samples were drawn for suspended respirable particulates, sulfur dioxide (SO_2), and nitrogen dioxide (NO_2).

Particulates were collected on a millipore filter (pore size, 0.8 µm) with the aid of an MSA cyclone sampler for respirable-size dust (particulates less than 7 µm in diameter). The filters, after being conditioned to standard temperature and humidity, were weighed to the nearest 10 µg before and after the sampling period. SO_2 and NO_2 were analyzed by accepted methods.[6]

Subjects carried the sampler with them for a 24-hour period on a day when they attended school. The sampler ran continuously and was taken to all activities including the walk to and from school, gym class, and meetings, sports, or any other after-school activities. At the end of the 24-hour sampling period, we recorded the daily activities, and details of the climatic conditions and the home environment, including the number of smokers in the home, the number of people and rooms in the home, the type of heating system used, the type of fuel used for cooking, the composition of the floors or floor coverings, the presence of open or closed windows, and the presence or absence of a fireplace and pets. Two subjects reported an atypical exposure or daily activity. In both of these situations the data were rejected and the sampling repeated.

In addition to the personal air sampling, outdoor sampling according to standard techniques[6] was conducted continuously as part of the community respiratory disease survey in Ansonia.[6] These outdoor measurements were taken

Table 1.—Personal Air Pollution Measurements by Race and Control or Disease Status, and by Presence of One or More Smokers in the Home

	No. of Subjects	Particulates (μg/m³)	SO_2 (μg/m³)	NO_2 (μg/m³)
Race				
Black				
Control	5	123 ± 33	5.7 ± 1.5	75 ± 18
Disease	5	96 ± 6.8	6.6 ± 2.1	58 ± 5.6
White				
Control	5	131 ± 9.8	4.9 ± 0.9	53 ± 12
Disease	5	108 ± 9.5	5.0 ± 1.2	59 ± 21
Smoker in home				
No	9	93 ± 13	5.7 ± 1.1	52 ± 9.3
Yes	11	132 ± 7.1*	5.4 ± 0.8	68 ± 11

NOTE: Measurements given as mean ± SE.
*$P < .05$; significant difference in mean particulate concentration between the two domestic smoking groups, using the *t*-test with the Cox-Cochran correction for unequal variances.[7]

within 2 miles of the homes and schools of the subjects under study. Subsequent to the personal monitoring, three subjects were studied with simultaneous indoor and outdoor particulate measurements. The personal air sampler was used in the home for indoor sampling, and a Hi-Vol sampler was used at a distance of from 4 to 6 meters from the home for outdoor sampling. These simultaneous comparisons were made over a 24-hour period.

The study was conducted from September through December 1973.

Results

Table 1 shows the mean personal air pollution measurements for all groups. The individual exposures varied widely, as reflected in the high standard error of the mean for most measurements. Compared to the Environmental Protection Agency air quality standards (EPAAQS),[6] these mean results were high for particulates, very low for SO_2, and moderately low for NO_2 (EPAAQS annual geometric means are 75 μg/m³, 80 μg/m³, and 100 μg/m³, for particulates, SO_2, and NO_2, respectively).

The mean particulate concentrations for subjects exposed to domestic cigarette or cigar smoke were significantly higher ($t' = 2.61$; $P < .05$ by the Cox-Cochran correction[7]) than those for subjects not so exposed (Table 1). Even in the latter group, particulate exposure was high, exceeding the EPAAQS of 75 μg/m³ for all but one subject, for whom particulates were 48 μg/m³. Exposure to NO_2 also tended to be greater in children exposed to domestic smoking. There was no significant difference between the two groups with respect to mean SO_2 exposure or pulmonary function, measured as the ratio of forced expiratory volume in 1 second to the forced vital capacity ($FEV_{1.0}$/FVC), and the ratio of the maximum expiratory flow at 50% of lung volume to the FVC (MEF50%/FVC).

Particulate exposure tended to be higher in subjects living in homes with a higher ratio of persons to number of rooms, but the association was not significant. No correlation was observed between pollutant levels and the other home factors recorded.

A comparison of disease and control groups showed higher particulate exposures among control subjects. However, this difference was not statistically significant and was reduced when the presence of domestic smoking was taken into account. There were no significant differences in pollutant exposures between black and white children.

Table 2.—Comparison of Personal and Outdoor Environmental Measurements

Pollutant	Concentration Levels (Mean ± SE) Personal*	Outdoor†
Particulates	114.5 ± 9.8	58.4 ± 5.9‡
SO_2	5.5 ± 0.7	12.0 ± 2.2§
NO_2	61.3 ± 7.2	100.1 ± 9.0//

NOTE: Data given in μg/m³.
*Data for all pollutants based on twenty measurements.
†Data for particulate concentration based on twenty-two measurements; data for SO_2 and NO_2 concentration based on twenty-one measurements.
‡$P < .001$. §$P < .05$. //$P < .01$: significant difference in mean pollutant concentration between the two groups, by Student's *t*-test and the *t*-test with the Cox-Cochran correction for unequal variances.[7]

Table 2 shows the average pollutant concentrations obtained with the personal sampler and those of the outdoor air measured in the same area during the 2-month study period. In-home measurements of particulate levels for three subjects living in homes with smokers were (in $\mu g/m^3$): subject 1, 112; subject 2, 57; and subject 3, 141. Simultaneous measurements taken outside the home for the same subjects 1, 2, and 3 were (in $\mu g/m^3$) 26, 48, and 61, respectively. Subjects 1 and 3 reported no respiratory symptoms, while subject 2 reported cough on most days.

Comment

We found that personal exposures to particulates were considerably higher than outdoor concentrations; in all but one subject, they exceeded the present annual EPAAQS. On one occasion, particulate exposure exceeded the maximum 24-hour concentration allowed not more than once per year (260 $\mu g/m^3$).[6] This is particularly interesting since the personal sampler is designed to measure only particulates less than 7 μm in diameter, while the outside and EPAAQS levels reflect particles in the range of 0.1-100 μm. On the average, personal SO_2 and NO_2 levels were well below outdoor levels and the EPAAQS. Previous investigations[8,9] have reported similar findings for NO_2, but not for particulates. Our results suggest the presence of an indoor source of particulates. Yocom et al.[10] also present data supporting the presence of indoor particulate and SO_2 sources.

The large variance among the individual exposures probably reflects true variation in personal air pollution loads. The subjects used in the study were cooperative, and the sampler was always locked to prevent interference. In general, the errors of measurement in the methods of pollutant analysis we used are smaller than the variations between subjects that we observed. However, variation in millipore filter weight with small changes in humidity may account for some of the variance noted in the particulate measurements. This problem can be overcome by using glass-fiber filters, which are less hygroscopic.

We could not find a relation between personal pollutant exposure and variations in climate. However, the study was conducted during a single season, and the indoor environment is probably protected from gross climatic influences.

We were able to demonstrate a significant relationship between particulate concentrations and one of the several [illegible] presence of at least one smoker in the subject's home was striking. Analysis of cigarette particulate emissions has shown that indoor smoking can lead to high particulate emissions.[11] An individual's exposure will depend on ventilation of the home and on his distance from a smoker. It has been suggested that cigarette smoking may cause increased NO_2 levels in a confined space.[12] NO_2 concentrations found in the homes of smokers tended to be higher than in the homes of nonsmokers. The ratio of persons to number of rooms in the home may also influence particulate exposure.

In general, pollutant exposure was similar for control and disease subjects. The significant difference observed for particulate expos[illegible] due to domestic smoking and was independent of the presence or absence of respiratory disease. We found no evidence to support a cause and effect relationship between pollutant exposure and respiratory disease.

Outdoor air measurements do not accurately reflect the air pollution load experienced by individuals who live in the area of sampling. Children probably spend more time outdoors than most adults; nevertheless, our analysis of reported daily activities showed that children are indoors from at least 60% to about 80% of an average school day. Therefore, indoor pollutant exposures, especially to cigarette smoke, are of particular interest. In this study, nineteen of the twenty children had a 24-hour particulate exposure higher than the annual mean of the primary air quality standard (75 $\mu g/m^3$), even though outdoor levels at this time were lower than the standard. Since a person's air pollutant load, especially for respirable particulates, appears to be determined primarily by indoor exposures, no significant improvement in respirable particulate loads can be expected to result from reduction of outdoor particulate levels, even in urban areas.

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